independent of the site of pacing. HRA pacing is associated with significant increase in incremental CD time over His, CSos and distal CS. Sustained AF was only initiated in 2 pts during HRA pacing with APBs.

Conclusion: 1) Local atrial CD in response to APBs is more prominent over His and CSos independent of the site of pacing. 2) HRA pacing is associated with significant dispersion of local atrial CD and may facilitate AF initiation. 3) LRA or HRA + distal CS pacing are effective in miniming the dispersion of local atrial CD.

1067-93 Left Atrial "Preexcitation" Is Not Useful in Preventing the Induction of Atrial Fibrillation from the High Right Atrium

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Background: Atrial Fibrillation (AF) could be initiated during sinus rhythm by shortly coupled premature beats originated in atrial regions with arrhythmogenic substrate as the vicinity of the sinus node or the crista terminalis.

Objectives: To simulate this circumstances at the EP lab and to assess with dual atrial stimulation (DAS): 1) If left atrial "preexcitation" is feasible and 2) If it could prevent AF induction.

Patients and Methods: In eight consecutive patients (P) aged 30.5 ± 12 years, 5 females, undergoing a EP study, at least 3 self-terminating episodes of AF were induced with one (3 P) two (4 P) or three (1P) atrial extrastimuli added to a 400 ms cycle length drive train delivered at the right atrial appendage (RAA). Only one P had clinically documented AF. After the reproducibly induction of AF, these P underwent DAS with identical numbers and coupling intervals of extrastimuli that induced the arrhythmia, but delivering the drive train simultaneously at the RAA and the distal coronary sinus (CS) with the extrastimuli being delivered only at the RAA.

Results: The development of AF could not be prevented with dual pacing in any patient despite a significant shortening of the interatrial conduction time of right atrial extrastimuli with this mode of pacing (the mean interatrial conduction interval of the last atrial extrastimulus was 187 ± 14 ms during RAA pacing versus 152 ± 19 ms during RAA-CS pacing, $p < 0.001$). The episodes of induced AF originated, in all but one P (in this P it was originated at the His bundle region), in the vicinity of the RAA. No statistically significant differences were noted in the characteristics of AF when comparing the episodes induced with or without dual atrial pacing.

Conclusion: In our model of AF originated at the RA, dual atrial pacing from the RAA and the CS, makes feasible to "preexcite" the left atrial activation, but does not prevent the initiation of AF.

1067-94 The Chronic Atrial Fibrillation Canine Model: A New Approach to Increase Yield and Efficiency

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Further investigation of atrial fibrillation (AF) depends on the use of an AF model which closely resembles chronic AF in humans. The best model currently available, the single-lead rapid atrial pacing dog model, has been documented to have a 6 week spontaneous AF yield of only 18%. We hypothesized that multi-site atrial pacing may increase the yield of chronic AF in a shorter time span. *Methods:* In 18 mongrel dogs (27–32 kg), pacemakers, (Pacesetter SX-2FAST) were implanted. In 5 dogs a single lead was fixed in the RA appendage (RAA), and in 13 dogs two leads were placed – one in the RAA and another in the low RA. In both groups, pacing was applied to the RAA at 400 B/M. In the second group, the low RA was paced at 130 B/M Surface and endocardial electrograms were examined weekly while pacing and with the pacemaker off. Echocardiographic assessment of cardiac chamber size and function was performed before and after completion of the pacing protocol. Atrial tissues were obtained for histological evaluation. *Results:* Among the 5 single-site pacing dogs, only 2 (40%) developed AF; this required pacing for 151 ± 124 days. All 13 of the dual-site pacing dogs (100%) developed spontaneous AF within 41 ± 19 days ($p < 0.05$ vs. single-site pacing). In 5 of these dogs, pacing was continued for 79 ± 21 days after the onset of AF. The pacemaker was then turned off for 38 ± 10 days during which the AF persisted. Echocardiography showed that the development of AF correlated with severe atrial enlargement and ventricular dysfunction. RA size increased from 20 ± 2 to 27 ± 5 mm, LA size increased from 32 ± 3 to 43 ± 9 mm, LVEF decreased from 50 ± 10% to 24 ± 7%. Development of AF was associated with atrial tissue changes such as fat infiltration, fibrosis, fibroelastosis, and myocardial disarray. Severity of the changes were mild in the first group versus moderate-severe in the second. *Conclusions:* 1) Dual-site pacing causes severe atrial myopathy more rapidly and with a higher yield than single-site pacing. 2) This model provides the morphological substrate for chronic AF, yielding a highly reproducible, efficient, and stable model of chronic AF. 3) Spontaneous persistence of AF makes this model similar to chronic AF in humans. 4) Development of chronic AF correlates with severe dilatation of both atria and ventricular dysfunction.

1067-95 Flecainide Versus Sotalol for Immediate Conversion of Atrial Fibrillation

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In a randomized study of 106 hemodynamically stable patients (pts) with atrial fibrillation (AF), intravenous flecainide (FLEC) was compared with intravenous sotalol (SOT) for immediate conversion to sinus rhythm. Pts with atrial flutter were not eligible. Trial medication was given by infusion over 15 minutes at a dose of 1.5 mg/kg body weight (maximum 150 mg). Exclusion criteria included significant left ventricular dysfunction, recent antiarrhythmic therapy and hypokalemia. Pts were stratified according to duration of AF (stratum I: ≥ 15 minutes to ≤ 24 hours; stratum II: > 24 hours to ≤ 7 days; stratum III: > 7 days to ≤ 6 months). Treatment was considered successful if stable sinus rhythm occurred within 2 hours of starting medication. Overall, conversion was achieved in 28 of 54 pts (52%) given FLEC and 12 of 52 pts (23%) given SOT ($p = 0.004$). Conversion rates of both drugs varied depending on duration of AF.

Duration of AF	Conversion rate (%)		p-value
	FLEC	SOT	
≥ 15 min to ≤ 24 hours	24/35 (69%)	11/36 (31%)	0.003
> 24 hours to < 7 days	4/ 9 (44%)	1/6 (17%)	NS
> 7 days to ≤ 6 months	0/10 (0%)	0/10 (0%)	NS
Total	28/54 (52%)	12/52 (23%)	0.004

Adverse effects were seen in 9 pts (17%) assigned to FLEC and in 10 pts (19%) assigned to SOT (including torsade de pointes in 1 pt) (p = NS).

Conclusions: Conversion to sinus rhythm within 2 hours by FLEC or SOT was limited to pts with AF of recent onset (≤ 7 days duration). At a dose of 1.5 mg/kg, FLEC is significantly more effective than SOT for immediate conversion of AF if the duration of the attack is less than 24 hours.

1067-96 Atrial Flutter, A Risk Factor for Stroke

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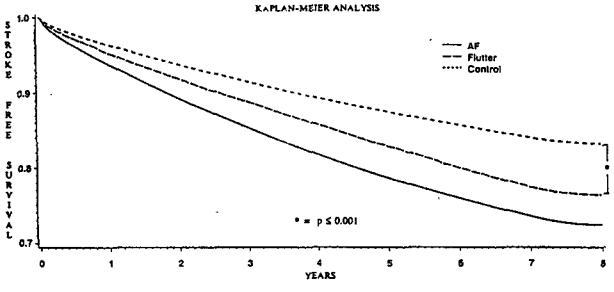
The embolic potential of atrial flutter in patients is not clear. We hypothesized that patients with atrial flutter are not at risk for stroke. Using a retrospective cohort study, we identified all Medicare patients hospitalized in 1984 with either a diagnosis of atrial fibrillation (N = 396,012) or atrial flutter (N = 20,209). A random sample of other hospitalized patients (N = 395,147) was used as the control group. The cohort was followed for 8-years. A Kaplan-Meier analysis was used to determine the likelihood of subsequent stroke. A Cox's proportional hazard model was used to determine the relative risk of stroke adjusting for age, race, sex, hypertension, diabetes and heart disease. *Results:* Patients with either atrial fibrillation or atrial flutter were at an increased risk for the development of stroke during the 8-year period. The relative risk for stroke in patients with atrial flutter was 1.4, $p \leq 0.001$ and in patients with atrial fibrillation was 1.6, $p \leq 0.001$.



Conclusion: Patients with either atrial flutter or atrial fibrillation are at an increased risk of stroke when compared to a hospitalized cohort.